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Derwent World Patents Index
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DATE: Wednesday, October 27, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB; PLUR=YES; OP=ADJ			
L10	L9 same l8	10	L10
L9	il-16	574	L9
L8	L7 same l6	168	L8
L7	rantes	1576	L7
L6	fibroblasts or fibroblast	18303	L6
DB=USPT; PLUR=YES; OP=ADJ			
L5	L2 same l1	10	L5
L4	L3 same l1	118	L4
L3	rantes	1040	L3
L2	il-16	113	L2
L1	fibroblasts or fibroblast	23619	L1

END OF SEARCH HISTORY

h e b b cg b e e ch

Set	Items	Description
S1	440480	FIBROBLASTS OR FIBROBLAST
S2	112474	AUTOANTIBODY OR AUTOANTIBODIES
S3	1591415	ACTIVATE OR ACTIVATED OR STIUMLATE OR STIMULATED
S4	1722215	ACTIVATE OR ACTIVATED OR STIMULATE OR STIMULATED
S5	119	S4 AND S2 AND S1
S6	79	RD (unique items)

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    1492303   ANTIBODY OR ANTIBODIES
S2     335599   FIBROBLASTS OR FIBROBLAST
S3      32717   S1 AND S2
S4      12038   S3/TI
S5    1239750   ACTIVATE OR ACTIVATION OR ACTIVATES
S6       3940   S5 AND S1 AND S2
S7       1819   S6/TI
S8     459282   S1/TI
S9     111004   S2/TI
S10      1089   S8 AND S9
S11    350271   S5/TI
S12        36   S11 AND S8 AND S9
S13      5132   S11 AND S8
S14      3960   FAP OR FIBROBLAST(W)ACTIVATION(W)PROTEIN
S15      5111   S13 NOT S14
S16      5111   S5 AND S15
S17      3940   S3 AND S2 AND S5
S18      1089   S8 AND S9
S19        76   S18 AND S5
S20        58   S19 NOT S14
S21        36   RD (unique items)
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Trying 31060000009998...Open

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                      *** DIALOG HOMEBASE(SM) Main Menu ***

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 service.  Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? b 410

       27oct04 11:14:39 User231886 Session D579.1
            $0.00    0.197 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.00  Estimated cost this search
     $0.00  Estimated total session cost   0.197 DialUnits

File 410:Chronolog(R) 1981-2004/Sept
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      Set  Items  Description
      ---  -----  -----------
? set hi ;set hi
HILIGHT set on as ''
HILIGHT set on as ''
? b 5 155 73 357 358 399
       27oct04 11:14:48 User231886 Session D579.2
            $0.00    0.097 DialUnits File410
     $0.00  Estimated cost File410
     $0.03  TELNET
     $0.03  Estimated cost this search
     $0.03  Estimated total session cost   0.293 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R) 1969-2004/Oct W3
         (c) 2004 BIOSIS
  File 155:MEDLINE(R) 1951-2004/Oct W4
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  File 357:Derwent Biotech Res. _1982-2004/Oct W5
         (c) 2004 Thomson Derwent & ISI
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         (c) 2004 DECHEMA
  File 399:CA SEARCH(R) 1967-2004/UD=14118
         (c) 2004 American Chemical Society
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      Set  Items  Description
      ---  -----  -----------
? s fibroblast or fibroblasts
          277489  FIBROBLAST
          276558  FIBROBLASTS
      S1  440480  FIBROBLAST OR FIBROBLASTS
? sa il(w)16
               0  A IL
         1331165  16
      S2       0  A IL(W)16
? s il(w)16
          392236  IL
         1331165  16
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      S3     873  IL(W)16
? s rantes
      S4   13405  RANTES
? s s2 and s3 and s4
               0  S2
             873  S3
           13405  S4
      S5       0  S2 AND S3 AND S4
? s s1 and s3 and s4
          440480  S1
             873  S3
           13405  S4
      S6      20  S1 AND S3 AND S4
? rd
...completed examining records
      S7      10  RD (unique items)
? t s7/7,k/all
>>>KWIC option is not available in file(s): 399

 7/7,K/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0014920113   BIOSIS NO.: 200400290870
Transforming growth factor-beta induces elevated interleukin-16 mRNA in
  synovial fibroblasts
AUTHOR: Aicher Wilhelm K (Reprint)
AUTHOR ADDRESS: Orthopaedic Surgery, UKT, Hoppe Seyler Str. 3, Tuebingen,
  BW, D 72076, Germany**Germany
AUTHOR E-MAIL ADDRESS: aicher@uni-tuebingen.de
JOURNAL: FASEB Journal  18 (4-5): pAbst. 779.9 2004 2004
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the
Genome  Washington, District of Columbia, USA  April 17-21, 2004; 20040417
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Introduction:Rheumatoid arthritis (RA) is a chronic inflammatory
  disease.   ***IL***  -  ***16***   is expressed at elevated levels in RA synovial
    ***fibroblasts***   (SF). Binding of   ***IL***  -  ***16***   to CD4 on
T-cells,
  macrophages or neutrophils induces chemotaxis and may activate these
  cells. As   ***IL***  -  ***16***   may contribute to the chronic inflammation RA
  we investigated mechanisms regulating IL-16
  expression.Methods:SF were prepared from synovial membranes of patients
  undergoing synovectomy after written consent. Cells were stimulated with
  cytokines including rhIL-1Beta, rhRANTES, rhTGF-Beta, rhTNF-a, rhVEGF and
    ***IL***  -  ***16***   expression was enumerated by quantitative RT-PCR.
  Results:VEGF or RANTES failed to modulate IL-16 mRNA
  responses in SF. Addition of TGF-Beta induced a statistically significant
    ***IL***  -  ***16***   response (194% +/- 65, p<0.006). Addition of IL-1Beta
  or TNF-a reduced   ***IL***  -  ***16***   mRNA amounts to 38.4% (+/-15, p<0.001)
  and 45% (+/- 35, p<0.0025). Conclusion:The data suggest that induction of
  IL-16 expression in SF is not associated with the most
  prominent pro-inflammatory cytokines promoting inflammation in RA,
  IL-1Beta and TNF-a. Further, as TGF-Beta is inducing a statistically
  significant and prominent IL-16 response, induction of
  IL-16 may represent an early event, possibly preceding
  infiltration of inflammatory mononuclear cells. 6418.
```

Transforming growth factor-beta induces elevated interleukin-16 mRNA in synovial **fibroblasts**
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DESCRIPTORS:
ORGANISMS: PARTS ETC: synovial **fibroblast**--

7/7,K/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014435434 BIOSIS NO.: 200300393864
Immunoglobulin activation of T cell chemoattractant expression in **fibroblasts** from patients with Graves' disease is mediated through the insulin-like growth factor I receptor pathway.
AUTHOR: Pritchard Jane; Han Rui; Horst Noah; Cruikshank William W; Smith Terry J (Reprint)
AUTHOR ADDRESS: Harbor-University of California-Los Angeles Medical Center, 1124 West Carson Street, Torrance, CA, 90502, USA**USA
AUTHOR E-MAIL ADDRESS: tjsmith@ucla.edu
JOURNAL: Journal of Immunology 170 (12): p6348-6354 June 15, 2003 2003
MEDIUM: print
ISSN: 0022-1767 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Graves' disease (GD) is associated with T cell infiltration, but the mechanism for lymphocyte trafficking has remained uncertain. We reported previously that **fibroblasts** from patients with GD express **IL-16**, a CD4-specific chemoattractant, and **RANTES**, a C-C chemokine, in response to GD-specific IgG (GD-IgG). We unexpectedly found that these responses result from a functional interaction between GD-IgG and the insulin-like growth factor (IGF)-I receptor (IGF-IR). IGF-I and the IGF-IR-specific IGF-I analog, des(1-3), mimic the effects of GD-IgG. Neither GD-IgG nor IGF-I activates chemoattractant expression in control ***fibroblasts*** from donors without GD. Interrupting IGF-IR function with specific receptor-blocking Abs or by transiently transfecting **fibroblasts** with a dominant negative mutant IGF-IR completely attenuates signaling provoked by GD-IgG. Moreover, GD-IgG displaces specific 125I-labeled IGF-I binding to **fibroblasts** and attenuates IGF-IR detection by flow cytometry. These findings identify a

novel disease mechanism involving a functional GD-IgG/IGF-IR bridge, which potentially explains T cell infiltration in GD. Interrupting this pathway may constitute a specific therapeutic strategy.

Immunoglobulin activation of T cell chemoattractant expression in **fibroblasts** from patients with Graves' disease is mediated through the insulin-like growth factor I receptor...

...ABSTRACT: cell infiltration, but the mechanism for lymphocyte trafficking has remained uncertain. We reported previously that **fibroblasts** from patients with GD express **IL-16**, a CD4-specific chemoattractant, and **RANTES**, a C-C chemokine, in response to GD-specific IgG (GD-IgG). We unexpectedly found...

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DESCRIPTORS:
...ORGANISMS: PARTS ETC: ***fibroblast***
CHEMICALS & BIOCHEMICALS: ***IL*** - ***16*** {interleukin-16...

... ***RANTES*** --

7/7,K/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014299638 BIOSIS NO.: 200300258282
Lung **fibroblasts** infected with respiratory syncytial virus express inflammatory and immunomodulatory mediators.
AUTHOR: Arnold Ralf (Reprint); Konig Wolfgang
AUTHOR ADDRESS: Institute of Medical Microbiology, Otto-von-Guericke-University, Leipziger Str. 44, Magdeburg, 39120, Germany**Germany
AUTHOR E-MAIL ADDRESS: ralf.arnold@medizin.uni-magdeburg.de; wolfgang.koenig@medizin.uni-magdeburg.de
JOURNAL: FASEB Journal 17 (4-5): pAbstract No. 161.22 March 2003 2003
MEDIUM: e-file
CONFERENCE/MEETING: FASEB Meeting on Experimental Biology: Translating the Genome San Diego, CA, USA April 11-15, 2003; 20030411
SPONSOR: FASEB
ISSN: 0892-6638 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: RSV is the most common cause of bronchiolitis and pneumonia among infants under 1 year of age. Lung ***fibroblasts*** are intimately engaged in the regulation of inflammatory lung responses. We hypothesized whether human lung **fibroblasts** (MRC-5, WI-38) are target cells for RSV infection. Our data show that both MRC-5 and WI-38 cells express the viral F-protein on their cell surface demonstrating the infection and synthesis of viral proteins. We observed a time-and RSV-dose dependent release of MIP-1alpha, **IL-16**, IL-8, **RANTES**, IL-6 and PGE2. In a Th1-cytokine environment (IFN-gamma) the RSV-infected WI-38 cells upregulated their MHC-II molecule expression. In contrast, the constitutive expression of MHC-I molecules was only moderately

upregulated within the first 24 h p.i. unlike to the adhesion molecule ICAM-1 which was significantly expressed on the cell surfaces of RSV-infected ***fibroblasts*** . Increased IL-6-, IL-8-, ***RANTES*** -and ICAM-1 mRNA steady state level were verified by RT-PCR, Taqman and mRNA-ELISA. By Western blot studies and inhibition experiments using specific antagonists an involvement of distinct signal transduction elements was demonstrated for the release of IL-8, ***RANTES*** and PGE2. These data suggest that lung **fibroblasts** are target cells for RSV infection and that they contribute to inflammatory as well as specific immune reponses by means of a variety of RSV-induced soluble and cell bound mediators. (BMBF-NBL3-01ZZ0107).

Lung **fibroblasts** infected with respiratory syncytial virus express inflammatory and immunomodulatory mediators.

...ABSTRACT: most common cause of bronchiolitis and pneumonia among infants under 1 year of age. Lung ***fibroblasts*** are intimately engaged in the regulation of inflammatory lung responses. We hypothesized whether human lung ***fibroblasts*** (MRC-5, WI-38) are target cells for RSV infection. Our data show that both...

...of viral proteins. We observed a time-and RSV-dose dependent release of MIP-1alpha, ***IL*** - ***16*** , IL-8, ***RANTES*** , IL-6 and PGE2. In a Th1-cytokine environment (IFN-gamma) the RSV-infected WI...

...adhesion molecule ICAM-1 which was significantly expressed on the cell surfaces of RSV-infected ***fibroblasts*** . Increased IL-6-, IL-8-, **RANTES**-and ICAM-1 mRNA steady state level were verified by RT-PCR, Taqman and mRNA...

...an involvement of distinct signal transduction elements was demonstrated for the release of IL-8, ***RANTES*** and PGE2. These data suggest that lung **fibroblasts** are target cells for RSV infection and that they contribute to inflammatory as well as...

DESCRIPTORS:
...ORGANISMS: human lung ***fibroblasts*** ; ...

...human lung ***fibroblasts***
ORGANISMS: PARTS ETC: lung **fibroblast**--
CHEMICALS & BIOCHEMICALS: ... ***IL*** - ***16*** {interleukin-16...

... ***RANTES*** ;

7/7,K/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013996412 BIOSIS NO.: 200200589923
Igs from patients with Graves' disease induce the expression of T cell chemoattractants in their **fibroblasts**
AUTHOR: Pritchard Jane; Horst Noah; Cruikshank William; Smith Terry J (Reprint)
AUTHOR ADDRESS: Department of Medicine, Division of Molecular Medicine, Harbor-University of California, Los Angeles Medical Center, 1124 West Carson Street, Building C-2, Torrance, CA, 90502, USA**USA
JOURNAL: Journal of Immunology 168 (2): p942-950 January 15, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Thyroid-associated ophthalmopathy and dermopathy are connective

tissue manifestations of Graves' disease (GD). Tissue remodeling is a prominent feature of both and is apparently driven by recruited T cells. In this study, we report that IgG isolated from patients with GD (GD-IgG) up-regulates T lymphocyte chemoattractant activity in GD-derived ***fibroblasts*** from orbit, thyroid, and several regions of skin. This chemoattractant activity, absent in **fibroblasts** from donors without known thyroid disease, is partially susceptible to neutralization by anti- ***IL*** - ***16*** and anti- ***RANTES*** Absolute ***IL*** - ***16*** is a CD4+-specific chemoattractant and ***RANTES*** is a C-C-type chemokine. **IL-16** and **RANTES** protein levels, as determined by specific ELISAs, are substantially increased by GD-IgG in GD ***fibroblasts*** . Addition of the macrolide, rapamycin, to **fibroblast** culture medium blocked the up-regulation by GD-IgG of **IL-16**, implicating the FRAP/mTOR/p70s6k pathway in the induction of ***IL*** - ***16*** expression. These findings suggest a specific mechanism for activation of **fibroblasts** in GD resulting in the recruitment of T cells. They may provide insight into a missing link between the glandular and extrathyroidal manifestations of GD.

Igs from patients with Graves' disease induce the expression of T cell chemoattractants in their **fibroblasts**

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DESCRIPTORS:
...ORGANISMS: PARTS ETC: dermal ***fibroblasts*** --
CHEMICALS & BIOCHEMICALS: ***IL*** - ***16*** {interleukin-16...

... ***RANTES*** {regulation upon activation normal T cell expressed and secreted

7/7,K/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013103555 BIOSIS NO.: 200100275394
Numerous growth factors, cytokines, and chemokines are secreted by human CD34+ cells, myeloblasts, erythroblasts, and megakaryoblasts and regulate normal hematopoiesis in an autocrine/paracrine manner
AUTHOR: Majka Marcin; Janowska-Wieczorek Anna; Ratajczak Janina; Ehrenman Karen; Pietrzkowski Zbigniew; Kowalska M Anna; Gewirtz Alan M; Emerson Stephen G; Ratajczak Mariusz Z (Reprint)
AUTHOR ADDRESS: Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine, University of Pennsylvania, 422 Curie Blvd, 405A Stellar Chance Labs, Philadelphia, PA, 19104, USA**USA
JOURNAL: Blood 97 (10): p3075-3085 May 15, 2001 2001
MEDIUM: print
ISSN: 0006-4971

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The aim of this study was to explore further the hypothesis that early stages of normal human hematopoiesis might be coregulated by autocrine/paracrine regulatory loops and by cross-talk among early hematopoietic cells. Highly purified normal human CD34+ cells and ex vivo expanded early colony-forming unit-granulocyte-macrophage (CFU-GM)-derived, burst forming unit-erythroid (BFU-E)-derived, and CFU-megakaryocyte (CFU-Meg)-derived cells were phenotyped for messenger RNA expression and protein secretion of various growth factors, cytokines, and chemokines to determine the biological significance of this secretion. Transcripts were found for numerous growth factors (kit ligand (KL), FLT3 ligand, **fibroblast** growth factor-2 (FGF-2), vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), insulinlike growth factor-1 (IGF-1), and thrombopoietin (TPO)); cytokines (tumor necrosis factor-alpha, Fas ligand, interferon alpha, interleukin 1 (IL-1), and **IL-16**); and chemokines (macrophage inflammatory protein-1alpha (MIP-1alpha), MIP-1beta, regulated upon activation, normal T cell expressed and secreted (**RANTES**), monocyte chemotactic protein-3 (MCP-3), MCP-4, IL-8, interferon-inducible protein-10, macrophage-derived chemokine (MDC), and platelet factor-4 (PF-4)) to be expressed by CD34+ cells. More importantly, the regulatory proteins VEGF, HGF, FGF-2, KL, FLT3 ligand, TPO, **IL-16**, IGF-1, transforming growth factor-beta1 (TGF-beta1), TGF-beta2, **RANTES**, MIP-1alpha, MIP-1beta, IL-8, and PF-4 were identified in media conditioned by these cells. Moreover, media conditioned by CD34+ cells were found to inhibit apoptosis and slightly stimulate the proliferation of other freshly isolated CD34+ cells; chemoattract CFU-GM-and CFU-Meg-derived cells as well as other CD34+ cells; and, finally, stimulate the proliferation of human endothelial cells. It was also demonstrated that these various hematopoietic growth factors, cytokines, and chemokines are expressed and secreted by CFU-GM-, CFU-Meg-, and BFU-E-derived cells. It is concluded that normal human CD34+ cells and hematopoietic precursors secrete numerous regulatory molecules that form the basis of intercellular cross-talk networks and regulate in an autocrine and/or a paracrine manner the various stages of normal human hematopoiesis.

...ABSTRACT: of this secretion. Transcripts were found for numerous growth factors (kit ligand (KL), FLT3 ligand, **fibroblast** growth factor-2 (FGF-2), vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), insulinlike...

...TPO)); cytokines (tumor necrosis factor-alpha, Fas ligand, interferon alpha, interleukin 1 (IL-1), and **IL-16**); and chemokines (macrophage inflammatory protein-1alpha (MIP-1alpha), MIP-1beta, regulated upon activation, normal T cell expressed and secreted (**RANTES**), monocyte chemotactic protein-3 (MCP-3), MCP-4, IL-8, interferon-inducible protein-10, macrophage...
...CD34+ cells. More importantly, the regulatory proteins VEGF, HGF, FGF-2, KL, FLT3 ligand, TPO, **IL-16**, IGF-1, transforming growth factor-beta1 (TGF-beta1), TGF-beta2, **RANTES**, MIP-1alpha, MIP-1beta, IL-8, and PF-4 were identified in media conditioned by...
...REGISTRY NUMBERS: ***fibroblast*** growth factor-2
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ***fibroblast*** growth factor-2...

7/7,K/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

16926686 PMID: 15322222

Synovial **fibroblasts** from patients with rheumatoid arthritis, like **fibroblasts** from Graves' disease, express high levels of **IL-16** when treated with Igs against insulin-like growth factor-1 receptor.

Pritchard Jane; Tsui Shanli; Horst Noah; Cruikshank William W; Smith Terry J

Division of Molecular Medicine, Harbor-University of California, Los Angeles Medical Center, Torrance, CA 90502, USA.

Journal of immunology (Baltimore, Md. - 1950) (United States) Sep 1 2004, 173 (5) p3564-9, ISSN 0022-1767 Journal Code: 2985117R

Contract/Grant Number: DK063121; DK; NIDDK; EY11708; EY; NEI; EY8976; EY; NEI; HL32802; HL; NHLBI; M01 RR00425; RR; NCRR

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We have reported recently that IgG from patients with Graves' disease (GD) can induce the expression of the CD4-specific T lymphocyte chemoattractant, **IL-16**, and **RANTES**, a C-C chemokine, in their ***fibroblasts*** . This induction is mediated through the insulin-like growth factor-1 receptor (IGF-1R) pathway. We now report that Abs from individuals with active rheumatoid arthritis (RA-IgG) stimulate in their synovial ***fibroblasts*** the expression of these same cytokines. IgG from individuals without known autoimmune disease fails to elicit this chemoattractant production. Furthermore, RA-IgG fails to induce ***IL*** - **16** or **RANTES** expression in synovial **fibroblasts** from donors with osteoarthritis. RA-IgG-provoked ***IL*** - ***16*** and **RANTES** production also appears to involve the IGF-1R because receptor-blocking Abs prevent the response. RA ***fibroblasts*** transfected with a dominant-negative mutant IGF-1R fail to respond to RA-IgG. IGF-1 and the IGF-1R-specific analog Des(1-3) also induce cytokine production in RA ***fibroblasts*** . RA-IgG-provoked ***IL*** - ***16***
expression is inhibited by rapamycin, a specific macrolide inhibitor of the Akt/FRAP/mammalian target of rapamycin/p70(s6k) pathway, and by dexamethasone. GD-IgG can also induce ***IL*** - ***16*** in RA **fibroblasts**, and RA-IgG shows similar activity in GD ***fibroblasts*** . Thus, IgGs from patients with RA, like those associated with GD, activate IGF-1R, and in so doing provoke T cell chemoattraction expression in **fibroblasts**, suggesting a potential common pathway in the two diseases. Immune-competent cell trafficking to synovial tissue is integral to the pathogenesis of RA. Recognition of this novel RA-IgG/ **fibroblast** interaction and its functional consequences may help identify therapeutic targets.

Record Date Created: 20040823

Record Date Completed: 20040921

Synovial **fibroblasts** from patients with rheumatoid arthritis, like **fibroblasts** from Graves' disease, express high levels of **IL-16** when treated with Igs against insulin-like growth factor-1 receptor.

... with Graves' disease (GD) can induce the expression of the CD4-specific T lymphocyte chemoattractant, **IL-16**, and ***RANTES*** , a C-C chemokine, in their ***fibroblasts*** . This induction is mediated through the insulin-like growth factor-1 receptor (IGF-1R) pathway...

... report that Abs from individuals with active rheumatoid arthritis (RA-IgG) stimulate in their synovial **fibroblasts** the expression of these same cytokines. IgG from individuals without known autoimmune disease fails to elicit this chemoattractant production. Furthermore, RA-IgG fails to induce **IL-16** or **RANTES** expression in synovial

fibroblasts from donors with osteoarthritis. RA-IgG-provoked ***IL***-16 and **RANTES** production also appears to involve the IGF-1R because receptor-blocking Abs prevent the response. RA ***fibroblasts*** transfected with a dominant-negative mutant IGF-1R fail to respond to RA-IgG. IGF...

... and the IGF-1R-specific analog Des(1-3) also induce cytokine production in RA ***fibroblasts*** . RA-IgG-provoked ***IL*** - ***16*** expression is inhibited by rapamycin, a specific macrolide inhibitor of the Akt/FRAP/mammalian target of rapamycin/p70(s6k) pathway, and by dexamethasone. GD-IgG can also induce ***IL*** - ***16*** in RA **fibroblasts**, and RA-IgG shows similar activity in GD ***fibroblasts*** . Thus, IgGs from patients with RA, like those associated with GD, activate IGF-1R, and in so doing provoke T cell chemoattraction expression in **fibroblasts**, suggesting a potential common pathway in the two diseases. Immune-competent cell trafficking to synovial tissue is integral to the pathogenesis of RA.. Recognition of this novel RA-IgG/**fibroblast** interaction and its functional consequences may help identify therapeutic targets.

; Arthritis, Rheumatoid--immunology--IM; Chemotactic Factors --biosynthesis--BI; **Fibroblasts**--drug effects--DE; **Fibroblasts** --immunology--IM; **Fibroblasts**--metabolism--ME; Graves' Disease --immunology--IM; Graves' Disease--metabolism--ME; Immunosuppressive Agents--pharmacology--PD; **RANTES**--metabolism--ME; Sirolimus--pharmaco logy--PD; Synovial Membrane--immunology--IM

Chemical Name: Antibodies; Chemotactic Factors; Immunosuppressive Agents; Interleukin-16; **RANTES**; Receptors, Somatomedin; Sirolimus

7/7,K/7 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

16082038 PMID: 14669949

The putative role of **fibroblasts** in the pathogenesis of Graves' disease: evidence for the involvement of the insulin-like growth factor-1 receptor in ***fibroblast*** activation.

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Autoimmunity (England) Sep-Nov 2003, 36 (6-7) p409-15, ISSN 0891-6934 Journal Code: 8900070

Contract/Grant Number: EY08976; EY; NEI; EY11708; EY; NEI

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Graves' disease when fully expressed affects the thyroid gland and connective tissues of the orbit and pretibium. While the glandular disease is relatively well-characterized, the pathogenesis of the orbital and dermal components remains enigmatic. In the following article, we review some of the evidence suggesting that **fibroblast** activation in Graves' disease might play an integral role in the tissue remodeling associated with ophthalmopathy. The thyrotropin receptor (TSHR) is expressed at low levels in several connective tissue depots and by their derivative ***fibroblasts*** , including those from the orbit. Little direct evidence currently links extra-thyroidal TSHR expression with Graves' disease. Very recent observations now implicate the insulin-like growth factor-1 receptor (IGF-1R) as a ***fibroblast*** activating antigen. When immunoglobulins from patients with the disease, with or without clinical ophthalmopathy, bind IGF-1R on the surface of **fibroblasts**, the receptor becomes activated and upregulates the expression of two T lymphocyte

chemoattractants, ***IL*** - ***16*** and ***RANTES*** . Thus, IGF-1R may
represent a second self-antigen with a pathogenic role in extra-thyroidal
Graves' disease. (43 Refs.)
Record Date Created: 20031212
Record Date Completed: 20040430

The putative role of **fibroblasts** in the pathogenesis of Graves' disease: evidence for the involvement of the insulin-like growth factor-1 receptor in ***fibroblast*** activation.
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IGF-1R may represent a second self-antigen with a pathogenic role in extra
...
Descriptors: Autoantigens; ***Fibroblasts***--physiology--PH; *Graves' Disease--physiopathology--PP; *Receptor, IGF Type 1--immunology--IM; Animals; Cytokines--physiology--PH; **Fibroblasts**--immunology--IM; Graves' Disease--immunology--IM; Orbit; Receptors, Thyrotropin--immunology --IM; Receptors, Thyrotropin--metabolism--ME

7/7,K/8 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10620624 PMID: 10725741
Cultured human **fibroblasts** express constitutive **IL-16** mRNA: cytokine induction of active **IL-16** protein synthesis through a caspase-3-dependent mechanism.
Sciaky D; Brazer W; Center D M; Cruikshank W W; Smith T J
Division of Molecular Medicine, Department of Medicine, Albany Medical College, Center, Albany, NY 12208, USA.
Journal of immunology (Baltimore, Md. - 1950) (UNITED STATES) Apr 1 2000, 164 (7) p3806-14, ISSN 0022-1767 Journal Code: 2985117R
Contract/Grant Number: EY08976; EY; NEI; EY11708; EY; NEI; HL32802; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Human **fibroblasts** can express numerous regulatory molecules that influence immune function. ***IL*** - ***16*** , a ligand for CD4, is a chemoattractant molecule expressed by lymphocytes, eosinophils, mast cells, and lung epithelium. It appears that the sole target for ***IL*** - ***16*** is the CD4-bearing cell. Here we demonstrate that ***fibroblasts*** from several tissues can express **IL-16** mRNA and protein as well as ***IL*** - ***16*** -dependent chemoattractant activity. The transcript is expressed abundantly under basal culture conditions as a 2.5-kb band on Northern analysis, similar to that observed in lymphocytes. ***IL*** - **16** protein and activity are undetectable in **fibroblast** cultures

under these same control conditions. However, when treated with proinflammatory cytokines such as IL-1beta, they express very high levels of **IL-16** protein and chemoattractant activity, a substantial component of which can be blocked with ***IL*** - ***16*** -neutralizing Absolute
The amount of **IL-16** protein released into the medium is 3- to 4-fold greater, on a per cell basis, than that observed in lymphocytes. The induction of **IL-16** protein by IL-1beta can be attenuated with specific inhibition of caspase-3, which could be detected in IL-1beta-treated ***fibroblasts*** . IL-1beta also induces ***RANTES*** mRNA, protein, and activity, and most of the chemoattractant activity released from **fibroblasts** not derived from **IL-16** can be attributed to ***RANTES*** . Human ***fibroblasts*** appear to be an important source of **IL-16** and through expression of this molecule may have key roles in the recruitment of CD4+ cells to sites of inflammation. ***IL*** - ***16*** expression and the mechanism involved in its regulation appear to be cell type specific.

Record Date Created: 20000504

Record Date Completed: 20000504

Cultured human **fibroblasts** express constitutive **IL-16** mRNA: cytokine induction of active **IL-16** protein synthesis through a caspase-3-dependent mechanism.

Human **fibroblasts** can express numerous regulatory molecules that influence immune function. ***IL*** - ***16*** , a ligand for CD4, is a chemoattractant molecule expressed by lymphocytes, eosinophils, mast cells, and lung epithelium. It appears that the sole target for ***IL*** - ***16*** is the CD4-bearing cell. Here we demonstrate that ***fibroblasts*** from several tissues can express **IL-16** mRNA and protein as well as ***IL*** - ***16*** -dependent chemoattractant activity. The transcript is expressed abundantly under basal culture conditions as a 2.5-kb band on Northern analysis, similar to that observed in lymphocytes. ***IL*** - **16** protein and activity are undetectable in **fibroblast** cultures under these same control conditions. However, when treated with proinflammatory cytokines such as IL-1beta, they express very high levels of **IL-16** protein and chemoattractant activity, a substantial component of which can be blocked with ***IL*** - ***16*** -neutralizing Absolute
The amount of **IL-16** protein released into the medium is 3- to 4-fold greater, on a per cell basis, than that observed in lymphocytes. The induction of **IL-16** protein by IL-1beta can be attenuated with specific inhibition of caspase-3, which could be detected in IL-1beta-treated ***fibroblasts*** . IL-1beta also induces ***RANTES*** mRNA, protein, and activity, and most of the chemoattractant activity released from **fibroblasts** not derived from **IL-16** can be attributed to ***RANTES*** . Human ***fibroblasts*** appear to be an important source of **IL-16** and through expression of this molecule may have key roles in the recruitment of CD4+ cells to sites of inflammation. ***IL*** - ***16*** expression and the mechanism involved in its regulation appear to be cell type specific.

Descriptors: Caspases--physiology--PH; *Cytokines--pharmacology--PD; *Fibroblasts--metabolism--ME; *Interleukin-16--biosynthesis--BI; *Interleukin-16--genetics--GE; *RNA, Messenger--biosynthesis--BI; Caspases--metabolism--ME; Cells, Cultured; Chemotaxis, Leukocyte--immunology--IM; Enzyme Activation--immunology--IM; **Fibroblasts**--enzymology--EN; **Fibroblasts**--immunology--IM; Inflammation Mediators--pharmacology--PD; Interleukin-1--pharmacology--PD; Lymphocytes--immunology--IM; Lymphokines--pharmacology...

7/7,K/9 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11782250 EMBASE No: 2002354689

Numerous growth factors, cytokines, and chemokines are secreted by human CD34SUP+ cells, myeloblasts, erythroblasts, and megakaryoblasts and regulate normal hematopoiesis in an autocrine/paracrine manner

Majka M.; Janowska-Wieczorek A.; Ratajczak J.; Ehrenman K.; Pietrzkowski Z.; Kowalska M.A.; Gewirtz A.M.; Emerson S.G.; Ratajczak M.Z.
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Blood (BLOOD) (United States) 15 MAY 2001, 97/10 (3075-3085)
CODEN: BLOOA ISSN: 0006-4971
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 65

The aim of this study was to explore further the hypothesis that early stages of normal human hematopoiesis might be coregulated by autocrine/paracrine regulatory loops and by cross-talk among early hematopoietic cells. Highly purified normal human CD34SUP+ cells and ex vivo expanded early colony-forming unit-granulocyte-macrophage (CFU-GM)-derived, burst forming unit-erythroid (BFU-E)-derived, and CFU-megakaryocyte (CFU-Meg)-derived cells were phenotyped for messenger RNA expression and protein secretion of various growth factors, cytokines, and chemokines to determine the biological significance of this secretion. Transcripts were found for numerous growth factors (kit ligand [KL], FLT3 ligand, **fibroblast** growth factor-2 [FGF-2], vascular endothelial growth factor [VEGF], hepatocyte growth factor [HGF], insulinlike growth factor-1 [IGF-1], and thrombopoietin [TPO]); cytokines (tumor necrosis factor-alpha, Fas ligand, interferon alpha, interleukin 1 [IL-1], and **IL-16**); and chemokines (macrophage inflammatory protein-1alpha [MIP-1alpha], MIP-1beta, regulated upon activation, normal T cell expressed and secreted [**RANTES**], monocyte chemotactic protein-3 [MCP-3], MCP-4, IL-8, interferon-inducible protein-10, macrophage-derived chemokine [MDC], and platelet factor-4 [PF-4]) to be expressed by CD34SUP+ cells. More importantly, the regulatory proteins VEGF, HGF, FGF-2, KL, FLT3 ligand, TPO, **IL-16**, IGF-1, transforming growth factor-beta1 (TGF-beta1), TGF-beta2, **RANTES**, MIP-1alpha, MIP-1beta, IL-8, and PF-4 were identified in media conditioned by these cells. Moreover, media conditioned by CD34SUP+ cells were found to inhibit apoptosis and slightly stimulate the proliferation of other freshly isolated CD34SUP+ cells; chemo-attract CFU-GM- and CFU-Meg-derived cells as well as other CD34SUP+ cells; and, finally, stimulate the proliferation of human endothelial cells. It was also demonstrated that these various hematopoietic growth factors, cytokines, and chemokines are expressed and secreted by CFU-GM-, CFU-Meg-, and BFU-E-derived cells. It is concluded that normal human CD34SUP+ cells and hematopoietic precursors secrete numerous regulatory molecules that form the basis of intercellular cross-talk networks and regulate in an autocrine and/or a paracrine manner the various stages of normal human hematopoiesis. (c) 2001 by The American Society of Hematology.

...of this secretion. Transcripts were found for numerous growth factors (kit ligand [KL], FLT3 ligand, **fibroblast** growth factor-2 [FGF-2], vascular endothelial growth factor [VEGF], hepatocyte growth factor [HGF], insulinlike...

...TPO]); cytokines (tumor necrosis factor-alpha, Fas ligand, interferon alpha, interleukin 1 [IL-1], and **IL-16**); and chemokines (macrophage inflammatory protein-1alpha [MIP-1alpha], MIP-1beta, regulated upon activation, normal T cell expressed and secreted [**RANTES**], monocyte chemotactic protein-3 [MCP-3], MCP-4, IL-8, interferon-inducible protein-10, macrophage...

...CD34SUP+ cells. More importantly, the regulatory proteins VEGF, HGF,

FGF-2, KL, FLT3 ligand, TPO, **IL-16**, IGF-1, transforming growth factor-beta1 (TGF-beta1), TGF-beta2, **RANTES**, MIP-1alpha, MIP-1beta, IL-8, and PF-4 were identified in media conditioned by...
DRUG DESCRIPTORS:
*stem cell factor; ***fibroblast** growth factor 2; *vasculotropin; * scatter factor; *somatomedin C; *thrombopoietin
...necrosis factor alpha; FAS ligand; alpha interferon; interleukin 1; interleukin 16; macrophage inflammatory protein 1; **RANTES**; monocyte chemotactic protein 3; monocyte chemotactic protein 4; thrombocyte factor 4 ; interleukin 8; gamma interferon...

7/7,K/10 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0347856 DBR Accession Number: 2004-20148 PATENT
Novel amended recombinant cell comprising one or more heterologous genes encoding chemokine or cytokine, useful for inducing/accelerating immune response in individual against immunogen - recombinant cell and cytokine and chemokine gene for use in disease therapy
AUTHOR: GAERTNER F H; LEE S L; SHUTTER R W
PATENT ASSIGNEE: GAERTNER F H; LEE S L; SHUTTER R W 2004
PATENT NUMBER: US 20040146484 PATENT DATE: 20040729 WPI ACCESSION NO.: 2004-552635 (200453)
PRIORITY APPLIC. NO.: US 681540 APPLIC. DATE: 20031007
NATIONAL APPLIC. NO.: US 681540 APPLIC. DATE: 20031007
LANGUAGE: English
ABSTRACT: DERWENT ABSTRACT: NOVELTY - An amended recombinant cell (ARC) (I) comprising one or more heterologous genes encoding a chemokine or a cytokine, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) inducing/accelerating (M1) an immune response in an individual against an antigen or immunogen, involves the step of administering (I) or a composition comprising (I), to an individual; (2) treating (M2) tumors, cancers, or malignancies, involves administering (I) or a composition comprising (I), to an individual; (3) inducing (M3) a desired biological effect in an individual, involves administering (I) or a composition comprising (I), to an individual; and (4) producing (I), involves introducing at least one heterologous gene encoding a cytokine, and optionally, a chemokine into a cell, growing the cell in a nutrient medium, harvesting the cells, and inactivating or fixing the cells. BIOTECHNOLOGY - Preferred Recombinant Cell: In (I), the heterologous gene encodes interleukin (IL)-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-15, **IL-16**, IL-18, IL-23, IL-24, erythropoietin, granulocyte colony stimulating factor (G-CSF), macrophage-CSF, platelet derived growth factor (PDGF), MSF, FLT-3 ligand, endothelial growth factor (EGF), **fibroblast** growth factor (FGF), aFGF(FGF-1), bFGF(FGF-2), FGF-3, FGF-4, FGF-5, FGF-6, FGF-7, insulin-like growth factor 1 (IGF-1), IGF-2, vascular endothelial growth factor (VEGF), interferon (IFN)-gamma, IFN-alpha, IFN-beta, leukemia inhibitor factor (LIF), ciliary neurotrophic factor (CNTF), oncostatin M, stem cell factor (SCF), transdermal growth factor (TGF)-alpha, TGF-beta1, THFbeta-2, chemokine chosen from BCA-i/BLC-1, BRAK/Kec, CXCL16, CXCR3, ENA-78/LIX, Eotaxin-1, Eotaxin-2/MPIF-2, Exodus-2/SLC, Fractalkine/Neurotactin, GROalpha/MGSA, HCC-1, I-TAC, Lymphotactin/ATAC/SCM, MCP-1/MCAF, MCP-3, MCP-4, MDC/STCP-1, ABCD-1, MIP-1alpha, MIP-1beta, MIP-2alpha/GRObeta, MIP-3alpha/Exodus/LARC, MIP-3beta/Exodus-3/ELC, MIP-4/PARC/DCCK1, PF-4, regulated upon activation, normal T-cell expressed and secreted (**RANTES**), SDF1alpha, TARC, and TECK, or 57 cytokines or chemokines such as TNF, IL, other growth and regulatory factors and CSF as given in the specification. The heterologous gene encodes IFN-gamma, where IFN-gamma is bovine, avian, fish or human, preferably bovine. The avian IFN-gamma is chicken IFN-gamma. (I)

further comprises a heterologous gene encoding IFN-alpha. The cell is a microbial cell of Gram-positive organisms, Gram-negative organism, yeast, or fungi, preferably Pseudomonas fluorescens. (I) further comprises a carrier. Preferred Method: (M1) further involves administration of an antigen of interest, preferably the administration of lipopolysaccharide (LPS). (I) co-express at least one antigen of interest. (M1) accelerates the development of immunoglobulin (Ig)-M, IgG, IgA, IgE, or IgY antibodies. (M1) further involves administering an antigen or immunogen prior to, concurrent with, or subsequent to the administration of composition comprising (I). (I) or composition comprising (I) has IFN-gamma and/or IFN-alpha. The IFN-alpha and IFN-gamma are of human, avian, bovine, or fish origin. The antigen or immunogen is a pathogen normally encountered by an individual in the environment or pathogenic substances specifically introduced into the environment of the individual. The antigen or immunogen is: biotoxin chosen from mycotoxins, trichothecene mycotoxin (T-2), Staphylococcal enterotoxin B, ricin, and Clostridium botulinum neurotoxin; a viral or bacterial pathogen chosen from smallpox, anthrax, Ebola virus, Yersinia pestis and weaponized microbial cells; or fungal pathogen. (M2) further involves administering chemotherapeutic agents, and optionally, tumor or cancer antigens. In (M3), the desired biological effect is chosen from activation or stimulation or macrophage in an individual, stimulation, suppression, or modulation of the immune system of an individual, increasing viral resistance in an individual, effecting a desired biological affect of cytokines, factors involved in immune response, chemokines, as given in the specification, treating shipping fever in animals, protecting the newborn animals from viral disease or bacterial gastroenteritis and reducing the severity of disease or disease symptoms. ACTIVITY - Cytostatic; Immunomodulator. No supporting data is given. MECHANISM OF ACTION - Immune response stimulator (claimed). USE - (I) is useful for inducing/accelerating an immune response in an individual against an antigen or immunogen, for treating tumors, cancers or malignancies, and for inducing a desired biological effect in an individual, where the desired biological effect is stimulation, suppression, or modulation of the immune system of an individual (all claimed). EXAMPLE - Completed Pseudomonas fluorescens fermentation culture was poured into a sterile beaker containing a sterilized magnetic stirring bar. The culture was stirred slowly, while the pH was monitored with an alcohol-sterilized pH-probe. Glacial acetic acid was added, drop-wise, for 10 minutes, until a pH of about 4.3 was reached. Following titration of the culture to about pH 4.3, concentrated 1% Lugol iodine (Lugol iodine; sterile distilled water, 90 mL; potassium iodide, 10 g/100 mL; iodine, 5 g/100 mL, glacial acetic acid, 10 mL). The solution was stirred well and aseptically transferred to a new, sterile beaker containing a sterile stirring bar. The solution was covered and stirred for one hour at room temperature. The cells were treated for longer periods of time (e.g., up to two hours) with similar results. The Lugol/cell mixture was transferred to a sterile 500 mL capped bottle and centrifuged at 7500 rpm for 15 minutes. The supernatant liquid was decanted and discarded. Sterile distilled water at room temperature was added up to the original volume, the pellet was dislodged with a sterile spatula, and the cells were re-suspended with an autoclave-sterilized, homogenized for about 10 seconds. Resuspension and centrifugation were repeated, as described above, three times to wash the cells free of Lugol solution. During the final wash the amended recombinant cells (ARC) were resuspended to 1/110 original volume and, frozen at -80degreesC in sterile screw-cap tubes for long-term storage. Thus, ARC were obtained. (34 pages)

...ABSTRACT: 5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-15, ***IL*** - **16** , IL-18, IL-23, IL-24, erythropoietin, granulocyte colony stimulating factor (G-CSF), macrophage-CSF, platelet derived growth factor (PDGF), MSF, FLT-3 ligand, endothelial growth factor (EGF), **fibroblast** growth factor (FGF), aFGF(FGF-1), bFGF(FGF-2), FGF-3,

FGF-4, FGF-5...

... MIP-4/PARC/DCCK1, PF-4, regulated upon activation, normal T-cell expressed and secreted (**RANTES**), SDF1alpha, TARC, and TECK, or 57 cytokines or chemokines such as TNF, IL, other growth...

?